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COVENANT
TO RESTRICT USE OF PROPERTY
Located At
241 Sixth Street
San Francisco, California

Recording Requested By:

The Knox Partners Limited Partnership
230 Fourth Street
San Francisco, CA 94124

When Recorded, Mail To:

Barbara Cook, Chief
Site Mitigation Branch
Department of Toxic Substances Control
700 Heinz Avenue, Suite 200
Berkeley, California 94710

This Covenant and Agreement ("Covenant") is made on the 15th day of June, 1994, by The Knox Partners Limited Partnership ("Covenantor"), which is the owner of record of certain property located at 241 Sixth Street, City and County of San Francisco, State of California, described in Exhibit "A" attached hereto and incorporated herein by this reference ("Property"), and by the California Department of Toxic Substances Control ("Department"), with reference to the following facts:

A. The Property consists of one parcel, identified as Lot 7A of Assessor's Block 3732, in the City and County of San Francisco, California. The Property has 8,000 square feet.

The Property is bordered to the north by Tehama Street, to the south by an electrical retail and repair shop, to the east by a condemned building, and to the west by Sixth Street. A map of the Property is attached hereto as Exhibit B.

B. The Property contains hazardous substances. The Property used to be marshlands which were filled in the late 1800's and early 1900's for development. The soil beneath the Property consists of fill and sandy silt. The soil concentrations for lead and zinc were above background levels. The soil concentrations for arsenic, carcinogenic PNAs, chromium VI, lead, and thallium exceeded the lowest total health-based levels and were selected as contaminants of concerns.

C. The hazardous substances and contaminants found on the Property are to be contained by the installation of a Cap (as described in the Cap Management Plan dated December 16, 1993, and approved by the Department) and the maintenance and monitoring of groundwater monitoring wells existing onsite. If this containment system were to be damaged by unauthorized excavation, breaching of the Cap, or impairment of the groundwater monitoring system, the occupants of the Property and nearby properties could be exposed to the contaminated soils. Exposures can take place via in-place contact, surface

water runoff and wind dispersal, resulting in dermal contact, inhalation, or ingestion by humans or animals. The purposes of the containment system and other mitigation measures are to eliminate any significant risks to human health and the environment. A description of potential human health and environmental effects of contaminants found on the Property is attached hereto as Exhibit C.

D. The Property is undergoing remediation under the supervision of the Department pursuant to a Voluntary Cleanup Agreement entered into between the Department and the Covenantor on or about January 19, 1994.

E. The Department has determined that deed restrictions need to be imposed on the Property to ensure full protection of public health and the environment.

F. The Property is presently owned by the Covenantor. The Property has been proposed for the development of affordable multi-family housing units.

G. Covenantor agrees that in order to protect the present and future public health and safety and the environment, the Property shall be used in such a manner as to avoid potential harm to persons or property which may result from any hazardous substance remaining on the Property.

ARTICLE I

GENERAL PROVISIONS

1.1 Provisions to Run With the Land. This Covenant sets forth protective provisions, covenants, restrictions, and conditions, (collectively referred to as "Restrictions"), upon and subject to which the Property and every portion thereof shall be improved, held, used, occupied, leased, sold, hypothecated, encumbered, and/or conveyed. Each and all of the Restrictions shall run with the land, and pass with each and every portion of the Property, and shall apply to and bind the respective successors in interest thereof. Each and all of the Restrictions are imposed upon the entire Property unless expressly stated as applicable to a specific portion of the Property. Each and all of the Restrictions are imposed pursuant to Section 25355.5 of the California Health and Safety Code and run with the land pursuant to said Section 25355.5. Each and all of the Restrictions are enforceable by the Department.

1.2 Concurrence of Owners Presumed. All purchasers, lessees, or possessors of any portion of the Property shall be deemed by their purchase, leasing, or possession of such Property, to be in accord with the foregoing and to agree for and among themselves, their heirs, successors, and assignees, and the agents, employees, and lessees of such owners, heirs, successors, and assignees, that the

Restrictions as herein established must be adhered to for the benefit of future owners and occupants and that their interest in the Property shall be subject to the Restrictions contained herein.

1.3 Incorporation Into Deeds and Leases. Covenantor agrees that the Restrictions set out herein shall be incorporated by reference in each and all deeds and leases of any portion of the Property.

ARTICLE II

DEFINITIONS

2.1 Cap. "Cap" shall mean the protective cover used to isolate contaminated soils on the Property from human or environmental exposure. The Cap has been constructed as outlined in Exhibit D attached hereto.

2.2 Department. "Department" shall mean the California State Department of Toxic Substances Control and shall include its successor agencies, if any.

2.3 Improvements. "Improvements" shall mean all buildings, structures, fixtures, roads, driveways, regradings, and paved parking areas, constructed or placed upon any portion of the Property.

2.4 Occupants. "Occupants" shall mean those persons entitled by ownership, leasehold, or other legal relationship to the exclusive right to occupy any portion of the Property.

2.5 Owner. "Owner" shall mean the Covenantor or its successors in interest, including heirs and assigns who hold title to all or any portion of the Property.

ARTICLE III

RESTRICTIONS

3.1 Restrictions on Use. Covenantor and Owner agree to restrict the use of the Property as follows:

3.1.1 The use of the Property is restricted to the development, construction, occupancy and maintenance of the affordable multi-family housing units as approved by the Department. No other use shall be allowed without the prior approval of the Department.

3.1.2 The Property shall not be used in such a way that will disturb or interfere with the integrity of any hazardous substance containment or monitoring system.

3.1.3 There shall not be any activity on the Property which will cause any potential harm to public health or

safety or the environment. F627583

3.2 Groundwater Monitoring. Covenantor and Owner shall perform and comply with the requirements of the Groundwater Monitoring Plan as approved by the Department for the remediation of the Property.

3.3 Operation and Maintenance of the Property. Covenantor and Owner shall perform and comply with the terms and requirements of the Operation and Maintenance Agreement to be entered into between the Department and Covenantor. In particular, Covenantor and Owner shall comply with the following requirements:

3.3.1 The Property shall be used and developed in such a way as to preserve the integrity of the Cap and the groundwater monitoring system installed on the Property.

3.3.2 Covenantor and Owner shall notify the Department of each of the following: (a) the type, cause, location and date of any disturbance to the Cap which could affect the ability of the Cap to contain subsurface hazardous substances on the Property, and (b) the type and date of repair of such disturbance. Notification to the Department and a request for any proposed earth moving or excavation shall be made

by telephone within 24 hours of the discovery of any Cap disturbance and by registered mail within five (5) days of both the discovery of Cap disturbance and the completion of required repairs.

3.3.3 The Department or its designated representatives shall have access to the Property for the purposes of inspection, surveillance, monitoring or other actions necessary to protect public health, safety or the environment.

3.4 Conveyance of Property. Covenantor and Owner shall provide a thirty(30)-day advance notice to the Department of any sale, lease, or other conveyance of the Property or an interest in the Property to a third person. The Department shall not have the authority to approve, disapprove, or otherwise affect any sale, lease, or other conveyance of the Property except as otherwise provided by law or by reason of this Covenant.

3.5 Enforcement. Failure of the Covenantor or Owner to comply with any of the Restrictions or requirements as set forth in this Covenant shall be grounds for the Department to require that the Covenantor or Owner modify or remove any Improvement constructed in violation of this Covenant. Any violation of the Covenant shall be grounds for the Department to take enforcement action, including

the filing of an administrative, civil or criminal action, as provided by law, against the Covenantor or Owner.

3.6 Notice in Agreements. Covenantor, Owner and Occupant shall execute a written instrument which shall accompany all purchase, lease, sublease, rental agreements, and other conveyance documents relating to the Property. The instrument shall contain the following statement:

> "The land described herein contains hazardous substances. Such condition renders the land, the property, and the owner, lessee, or other occupant of the land or property subject to the requirements, restrictions, provisions, and liabilities contained in Chapter 6.5 and Chapter 6.8 of Division 20 of the California Health and Safety Code. This statement is not a declaration that a hazard exists".

ARTICLE IV

VARIANCE AND REMOVAL OF RESTRICTIONS

4.1 Variance. Any Owner or, with the Owner's consent, any Occupant of the Property or any portion thereof, may apply to the Department for a written variance from any of the Restrictions or

requirements of this Covenant. Such application shall be made in accordance with Section 25233 of the California Health and Safety Code.

4.2 Removal of Restrictions. Any Owner or, with the Owner's consent, any Occupant of the Property or a portion thereof, may apply to the Department to remove any of the Restrictions or requirements of this Covenant as they apply to all or any portion of the Property. Such application shall be made in accordance with Section 25234 of the California Health and Safety Code.

4.3 Term. Unless modified or removed in accordance with Section 4.1 or Section 4.2 above, the Restrictions and requirements of this Covenant shall continue in effect in perpetuity.

ARTICLE V

MISCELLANEOUS

5.1 No Dedication Intended. Nothing set forth herein shall be construed to be a gift or dedication, or offer of a gift or dedication, of the Property or any portion thereof, to the general public for any purposes.

5.2 Notices. Whenever any person gives or serves any notice, demand, or other communication with respect to this Covenant, such notice, demand, or communication shall be in writing and shall be sent simultaneously to an authorized representative of the Covenantor (or Owner) and to the Department, in certified mail with return receipt requested.

5.3 Partial Invalidity. If any portion of this Covenant is determined to be invalid or unenforceable for any reason, the remaining portion of this Covenant shall remain in full force and effect.

5.4 Recordation. This Covenant shall be executed by the Covenantor and by the Department. This Covenant shall be recorded by the Covenantor in the San Francisco County Recorder's Office within ten (10) days of the date of execution as set forth above.

IN WITNESS THEREOF, the Covenantor and the Department execute this Covenant as of the date set forth above.

"Covenantor"

THE KNOX PARTNERS LIMITED PARTNERSHIP

By: GP/TODCO, Inc.
MANAGING GENERAL PARTNER

By: _____
John Elberling
Executive Vice-President
GP/TODCO, Inc.

"Department"

Barbara J Cook

Barbara Cook, Chief
Site Mitigation Branch
Department of Toxic Substances Control

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State of CALIFORNIA
County of ALAMEDA

On JUNE 15, 1994 (DATE) before me, V.K. CHOPRA NOTARY PUBLIC (NAME, TITLE OF OFFICER - I.E., "JANE DOE, NOTARY PUBLIC")

personally appeared BARBARA JEAN COOK AND JOHN HENRY ELBERLING (NAME(S) OF SIGNER(S))

☐ personally known to me - OR - ☐ proved to me on the basis of satisfactory evidence to be the person(s) whose name(s) is/are subscribed to the within instrument and acknowledged to me that he/she/they executed the same in his/her/their authorized capacity(ies), and that by his/her/their signature(s) on the instrument the person(s), or the entity upon behalf of which the person(s) acted, executed the instrument.

Witness my hand and official seal.

V. K. CHOPRA
Comm. #973228
NOTARY PUBLIC - CALIFORNIA
CONTRA COSTA COUNTY
Comm. Expires Sept. 13, 1996

(SEAL)

(SIGNATURE OF NOTARY)

RIGHT THUMBPRINT (OPTIONAL)

TOP OF THUMB HERE

CAPACITY CLAIMED BY SIGNER(S)

☐ INDIVIDUAL(S)
☐ CORPORATE _____
OFFICER(S) _____ (TITLE(S))
☐ PARTNER(S)
☐ ATTORNEY IN FACT
☐ TRUSTEE(S)
☐ GUARDIAN/CONSERVATOR
☐ OTHER: _____

SIGNER IS REPRESENTING:
(NAME OF PERSON(S) OR ENTITY(IES))

ATTENTION NOTARY: The information requested below is **OPTIONAL**. It could, however, prevent fraudulent attachment of this certificate to any unauthorized document.

THIS CERTIFICATE MUST BE ATTACHED TO THE DOCUMENT DESCRIBED AT RIGHT:

Title or Type of Document _____
Number of Pages _____ Date of Document _____
Signer(s) Other Than Named Above _____

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EXHIBIT A

241-6th Street Site
San Francisco, California

Legal description of the above site

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EXHIBIT A

241-6th Street Site
San Francisco, California

THE LAND REFERRED TO IN THIS POLICY IS SITUATED IN THE CITY AND COUNTY OF SAN FRANCISCO, STATE OF CALIFORNIA, AND IS DESCRIBED AS FOLLOWS:

COMMENCING AT THE POINT OF INTERSECTION OF THE NORTHEASTERLY LINE OF 6TH STREET; WITH THE SOUTHEASTERLY LINE OF TEHAMA STREET; AND RUNNING THENCE SOUTHEASTERLY ALONG SIDE LINE OF 6TH STREET 80 FEET; THENCE AT A RIGHT ANGLE NORTHEASTERLY 100 FEET; THENCE AT A RIGHT ANGLE OF SOUTHEASTERLY ALONG SIDE LINE OF TEHAMA STREET 100 FEET TO THE POINT OF COMMENCEMENT.

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EXHIBIT B

241-6th Street Site
San Francisco, California

A Map of the Property

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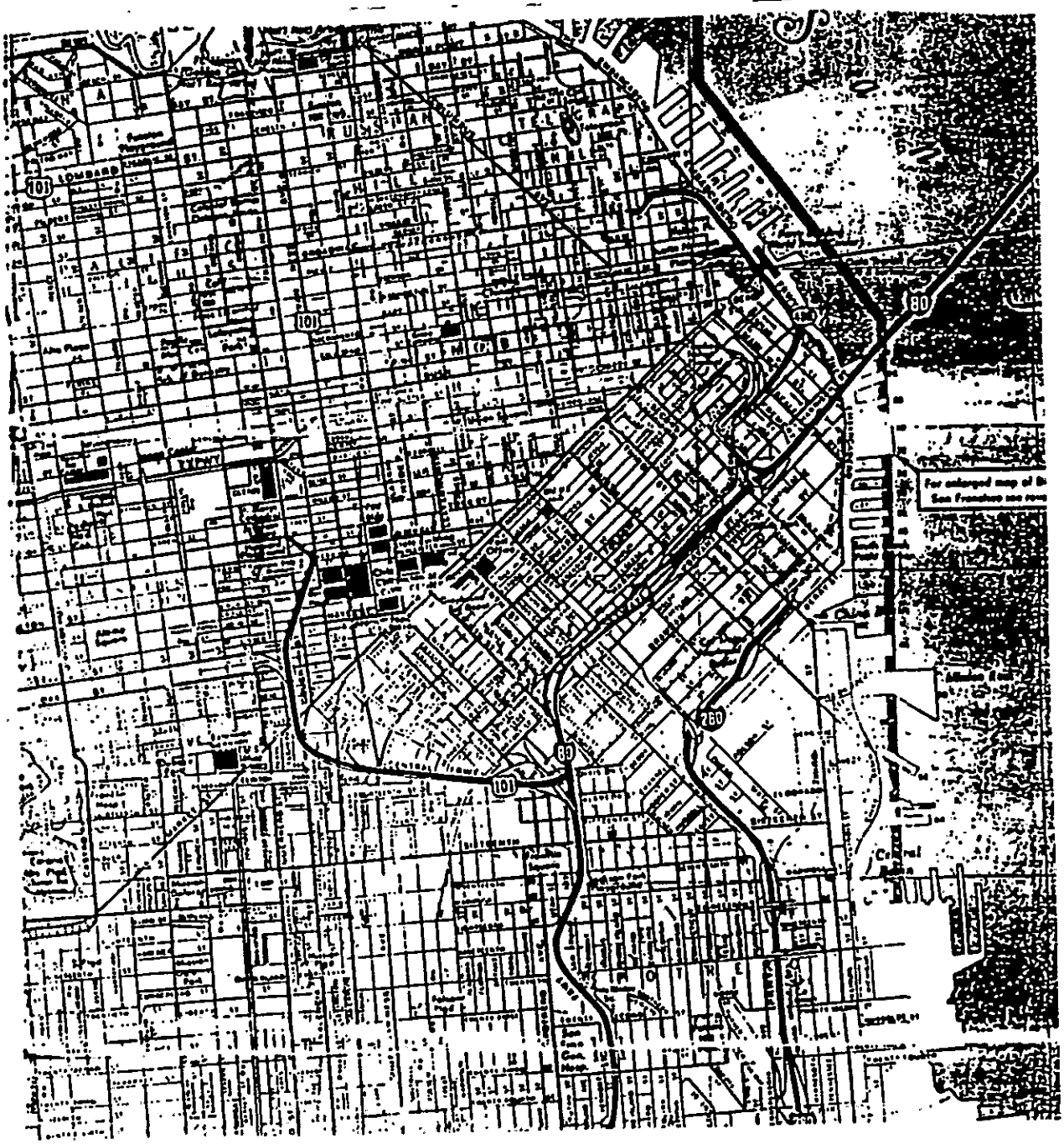

Scale: As Shown	April 15, 1994	**SITE LOCATION** **241 Sixth Street** **San Francisco, California**	MAP
Applied Remedial Services, Inc.			Project No. 3041

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EXHIBIT C

241-6th Street Site
San Francisco, California

A description of potential human health and the environmental effects of contaminants found on the property.

TOXICITY PROFILE

ARSENIC

Introduction

Arsenic is a silver-gray, brittle, crystalline metal. Its prevalence in the environment is due to both natural and anthropogenic sources. Arsenic is a naturally occurring substance in the earth's crust and is found widely in nature as arsenopyrite (*International Labour Organization, 1983*). Ceramics manufacturing, copper smelting industries, and pesticides are anthropogenic sources of arsenic in the environment (*ATSDR, 1989*). Oral exposure to inorganic arsenic primarily affects the skin and nervous system, and inhalation exposure affects the lung. Further details of arsenic toxicity are presented below.

Developmental/Reproductive Toxicity

Epidemiological studies found an increased abortion rate in women occupationally exposed via inhalation to arsenic fumes; decreased birth weight was also seen in babies born to similarly exposed women, although the data were not adequate to implicate arsenic as the causative agent (*Nordstrom et al., 1978a-d; ATSDR, 1989*). Most information on developmental toxicity is from animal studies, which have shown that arsenite (As^{+3}) can cause fetal malformations in rodents but arsenate (As^{+5}) only causes decreased birth weight and an increased abortion rate (*ATSDR, 1989*). No human reproductive data were located in the available scientific literature; in animals orally exposed to arsenite a small decrease in average litter size and an increase in the ratio of males to females were observed (*Schroeder and Mitchener, 1971*).

Noncarcinogenic Effects

Acute Toxicity

Severe gastrointestinal damage with nausea, vomiting, and diarrhea can occur following oral exposure in humans. Intense thirst, pharyngeal edema, and abdominal pain have also been observed. Irritation of the skin, eyes, nasal mucosa, pharynx, and bronchi may develop following exposure to airborne arsenic. Ingestion of very high doses may produce acute encephalopathy (*ATSDR, 1989*).

Chronic Toxicity

Chronic oral exposure to arsenic in humans results primarily in skin lesions and peripheral neuropathies with possible vascular disease. Skin lesions are very prevalent with chronic exposures and consist of hyperkeratosis and hyperpigmentation, usually in areas of the body not normally exposed to sunshine. Peripheral neuropathies involve both sensory and motor pathways; paresthesia, hyperesthesia, neuralgia, muscle pain, and weakness are typical manifestations (*ATSDR, 1989*). Blackfoot disease, a peripheral vascular disease characterized by gangrene of the extremities, has also been attributed to chronic ingestion exposure to arsenic; however, there is some question as to whether Blackfoot disease is strictly due to arsenic exposure (*Lu, 1990*).

Repeated oral exposure to arsenic in humans has also been observed to result in hematopoietic effects (anemia, leukopenia, eosinophilia), cardiovascular effects (myocardial infarction, arterial thickening), hepatic effects (necrosis, fatty changes, cirrhosis), and renal effects (hematuria) (*ATSDR 1989*).

The EPA-reported subchronic and chronic oral reference doses (sRfD and cRfD) for inorganic arsenic are both 3×10^{-4} mg/kg/day (*EPA, 1992a,b*). These values were based on epidemiologic studies performed by Tseng et al. (*1968*) and Tseng (*1977*) who calculated a no-observed adverse effect level (NOAEL) of 0.009 mg/l

(0.0008 mg/kg/day) and a lowest-observed adverse effect level (LOAEL) of 0.17 mg/l (0.014 mg/kg/day) for hyperpigmentation, keratosis, and vascular complications. An uncertainty factor of 3 was used to account for a lack of data that would preclude reproductive toxicity as a critical effect and for some uncertainty as to whether the NOAEL addressed all sensitive individuals (*EPA, 1992b*). On the basis of the information available, the EPA assigned a "medium" confidence level to the oral RfD on the basis of medium confidence in the study because doses were not well characterized and other contaminants were present, and medium confidence in the database because there were problems with the available epidemiological studies (*EPA, 1992b*).

Chronic inhalation exposure results in a toxicity profile similar to that for chronic oral exposure with additional effects on the respiratory system, including pulmonary insufficiency, tracheobronchitis, and perforation of the nasal septum. Cirrhosis of the liver and portal hypertension are also attributed to chronic inhalation exposure to arsenic. The EPA has not established subchronic or chronic inhalation RfDs due to a lack of adequate data (*EPA, 1992b*).

Carcinogenic Effects

Chronic oral exposure to arsenic can result in skin cancer; both squamous and basal cell carcinomas have been observed. Various studies have found widely different latency periods for the development of these cancers, ranging from 6 to 50 years. The most reasonable estimate of the average latency period for skin cancer is 24 years (*EPA, 1988*). Some evidence suggests that ingestion of arsenic may also contribute to lung cancer as well as cancers of the bladder, kidney, liver, and colon (*EPA, 1988*). The EPA-reported oral unit risk is 5×10^{-5} $(mg/l)^{-1}$; the equivalent oral slope factor is 1.75 $(mg/kg/day)^{-1}$ (*EPA, 1988*). This value is based on the epidemiologic study by Tseng

(*1977*), who reported an increased prevalence of skin cancer following exposure to arsenic in drinking water (*EPA, 1992b*).

Chronic inhalation exposure to arsenic results in an increased incidence of lung cancer. The range of reported latency periods associated with the development of these cancers is 13 to 50 years with an average of 31 years. Liver cancer has also been reported to occur following chronic inhalation of arsenic (*EPA, 1992b*). The EPA-reported inhalation unit risk value is 4.3×10^{-3} $(\mu g/m^3)^{-1}$, which converts to an inhalation slope factor of 15.1 $(mg/kg/day)^{-1}$ (*EPA, 1992b*). This slope factor is based on the epidemiologic studies of Brown and Chu (*1983a,b,c*), Lee-Feldstein (*1983*), Higgins (*1982*), and Enterline and Marsh (*1982*). These investigations evaluated male occupational exposure via inhalation of arsenic at the ASARCO and Anaconda Smelters, which resulted in statistically significant increases in the incidence of lung cancer. Based on the available scientific information, the EPA classified arsenic as a Group A chemical (known human carcinogen) via both the oral and inhalation exposure routes (*EPA, 1992b*).

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1989. *Toxicological Profile for Arsenic.* U.S. Public Health Service. March.

Brown, C.C., and K.C. Chu., 1983a. Approaches to Epidemiologic Analysis of Prospective and Retrospective Studies: Example of Lung Cancer and Exposure to Arsenic. In *Risk Assessment Proc. SIMS Conf. on Environ. Epidemiol.* June 28-July 2, 1982, Alta, VT. SIAM Publication. Cited in EPA, 1992b.

_____, 1983b. Implications of the Multistage Theory of Carcinogenesis Applies to Occupational Arsenic Exposure. *J.Natl. Cancer Inst.* 70:455-463.

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TOXICITY PROFILE

CARCINOGENIC PNAs

Introduction

Polycyclic aromatic hydrocarbons (PNAs) can be divided into two groups based on their toxicity: carcinogenic and noncarcinogenic. The following information is based on benzo(a)pyrene (BaP), the most widely studied of the potentially carcinogenic PNAs (carcinogenic cPNAs). BaP is the only carcinogenic PNA with established toxicity values (*EPA, 1992a,b*). Other carcinogenic PNAs are discussed briefly on the basis of a toxicity equivalence scheme by which the carcinogenicity of the other carcinogenic PNAs is numerically compared to the carcinogenicity of BaP.

Benzo(a)pyrene

BaP is a pale yellow chemical that occurs naturally in fossil fuels. Anthropogenic sources of BaP in the environment include exhaust from motor vehicles and other gasoline and diesel engines, emissions from coal-, oil-, and wood-burning stoves and furnaces, cigarette smoke, and general soot and smoke of industrial, municipal, and domestic origins (*ATSDR, 1990c*). Target sites of BaP toxicity include the bone marrow, reproductive organs, gastrointestinal tract, and respiratory system. Further details of BaP toxicity are presented below.

Developmental/Reproductive Toxicity

No studies on the developmental or reproductive effects of BaP in humans could be found in the available literature; no animal data on inhalation or dermal exposures were located. Oral exposure studies in animals indicated that *in utero* exposure to BaP is

associated with developmental toxicity and adverse reproductive effects. Investigators have reported a decreased fertility index, a high incidence of sterility in progeny, an increased incidence of stillbirths, altered gonadal development in offspring, and an increased incidence of malformations at birth following inhalation exposure of the dams (mothers) to BaP (*ATSDR, 1990c*).

Noncarcinogenic Effects

Acute Toxicity

Data on acute toxicity in humans and acute inhalation toxicity of BaP in animals were not located in the available scientific literature. Short-term oral exposure in animals has been shown to result in death due to bone marrow depression (*ATSDR, 1990c*).

Chronic Toxicity

Few data on noncarcinogenic effects of chronic BaP exposure in animals and no human data were located in the literature. Mice subchronically exposed via ingestion exhibited bone marrow depression with subsequent hemorrhage or infection (*ATSDR, 1990c*). The EPA has not established oral and inhalation reference doses (RfDs) for BaP (*EPA, 1992a*).

Carcinogenic Effects

The carcinogenicity of BaP has been tested extensively in animals. BaP has been demonstrated to be both a local and a systemic carcinogen, producing tumors at the point of exposure (*EPA, 1992b*). When ingested, BaP has induced an increased incidence of upper digestive tract tumors, stomach tumors, lung adenomas, and leukemias in animals. Animals exposed to BaP via inhalation developed tumors of the nasal cavity,

larynx, trachea, and pharynx (*ATSDR, 1990c, EPA, 1984a*). Intratracheal administration of BaP has resulted in an increased incidence of respiratory tract neoplasms and lung adenomas (*EPA, 1992a*). BaP has also been shown to induce skin tumors in animals following dermal exposure (*ATSDR, 1990c*).

Epidemiological studies have shown an association between skin contact with carcinogenic PNAs containing BaP and an increased risk of skin cancer. Lung cancer has been shown to be induced in humans by various mixtures of carcinogenic PNAs known to contain BaP including cigarette smoke, roofing tar, and coke-oven emissions. These studies are, however, insufficient to unequivocally correlate BaP exposure with carcinogenicity in humans (*ATSDR, 1990c*). The EPA has classified BaP as a Group B2 chemical or probable human carcinogen. The established slope factors for BaP are 5.79 and 6.1 $(mg/kg/day)^{-1}$ via the oral and inhalation routes of exposure, respectively (*EPA, 1992a,b*).

Other Carcinogenic PNAs

The following sections discuss the toxicity of other carcinogenic PNAs including (*Chu and Chen, 1984*) -

- Benzo(a)anthracene
- Benzo(b)fluoranthene
- Chrysene
- Dibenzo(a,h)anthracene
- Indeno(1,2,3-cd)pyrene.

Developmental/Reproductive Toxicity

Rats administered dibenzo(a,h)anthracene via subcutaneous injection daily from the first day of pregnancy exhibited an increased incidence of fetal death and resorption and possible long-term effects on fertility (*Wolfe and Bryan, 1939*). Parenteral administration of chrysene to mice in the perinatal period resulted in an increased incidence of hepatic tumors (*Grover et al., 1975; Buening et al., 1979*). No data could be located in the available scientific literature on the potential developmental or reproductive effects of carcinogenic PNAs on humans, and no other animal data on these or other carcinogenic PNAs could be located.

Noncarcinogenic Effects

Acute oral exposure to coal tar, a material that contains numerous carcinogenic PNAs, has been found to cause severe liver damage in pigs; however, acute oral and dermal administration of many carcinogenic PNAs or carcinogenic PNA mixtures have been shown to result in only mild toxicity in test organisms. Other carcinogenic PNAs, once isolated from the mixture, can cause specific, more severe symptoms including severe eye and skin irritation and damage to the liver, kidneys, lungs, and central nervous system (*EPA, 1984b*).

Repeated application of the potentially carcinogenic PNAs to the skin can cause dermatitis, folliculitis, photosensitization, and cancer. Additional data on the nontumor-related chronic toxicity of carcinogenic PNA-containing mixtures could not be found in the available literature.

Carcinogenic Effects

The carcinogenicity of PNAs as a class of chemicals has been studied extensively. Certain PNAs are considered to be carcinogenic when administered by all routes of

exposure; however, not all carcinogenic PNAs are carcinogens. Some including anthracene, acenaphthylene, acenaphthene, fluorene, fluoranthene, naphthalene, and phenanthrene show either no evidence or equivocal evidence of carcinogenicity (see the toxicity profile on noncarcinogenic carcinogenic PNAs based on naphthalene). One of the most extensively studied carcinogenic PNAs is BaP, which is clearly carcinogenic by all routes of exposure in animals. Other strong animal carcinogens of the PNA class include 7,12-dimethylbenz(a)anthracene, dibenzo(a,h)anthracene, 3-methylcholanthrene, 5-methylchrysene and dibenz(a,h)acridine (*EPA. 1984b*).

Interestingly, there is not yet clear evidence that individual PNAs are carcinogenic to humans although the carcinogenicity is inferred from animal studies. On the other hand, mixtures containing a variety of carcinogenic PNAs have long been suspected of causing cancer in humans. For example, soot and coal tar were first suspected in the late eighteenth century to be carcinogenic, which was later confirmed by experimental animal studies. In addition, these compounds have also been found to contribute to the human carcinogenicity of cigarette smoke. Epidemiological studies of various worker populations have shown a clear association between exposure to PNA-containing mixtures and increased cancer risk (*ATSDR. 1990c*). Currently, the EPA has classified many of the above carcinogenic PNAs as Group B2 chemicals or probable human carcinogens. It must be emphasized that a variety of factors play important roles in determining the carcinogenic potency of specific PNAs. These include but are not limited to structure, cellular transport, storage potential, enzyme inducibility, oxidative metabolism, and rate of excretion.

PNAs have also been studied extensively in short-term mutagenicity assays. The same principles apply to these results as to those for carcinogenic effects, i.e., many

PNAs are negative and many are strongly positive in mutagenicity assays. In general, those PNAs that are strongly carcinogenic also appear to be mutagenic.

Toxicity Equivalence Factors

To quantitatively assess the carcinogenic risk of a mixture of PNAs, two methods have been suggested. The first involves assessing all carcinogenic PNAs as if they had the same carcinogenic potency as BaP. This approach was recommended by the EPA (*1984; 1988*) and is regarded as a highly conservative method that may overestimate carcinogenicity because available data, although limited, suggest that some carcinogenic PNAs are significantly less potent than BaP. The EPA is currently reviewing alternative toxicity equivalency policies, which include the draft document issued by ICF-Clement (*1988*). The Department of Toxic Substances Control of the California Environmental Protection Agency (Cal-EPA) advocates the use of the current EPA policy for a baseline quantitative risk assessment for carcinogenic PNAs (i.e., all carcinogenic PNAs are equivalent to BaP in potency) and, in addition, suggests the use of a toxic equivalence approach as an alternative (*DHS, 1990*).

This relative potency methodology may significantly reduce the conservatism in evaluating the toxicity of the carcinogenic PNAs. The following toxic equivalence factors (TEFs), based on the data of Chu and Chen (*1984*), can also be used because other relative potency reports are controversial and still in draft form:

Compound	TEF
Benzo(a)anthracene	0.0134
Benzo(a)pyrene	1.0
Benzo(b)fluorene	0.08
Benzo(k)fluoranthene	0.0044
Chrysene	0.0012
Dibenzo(a,h)anthracene	0.69
Indeno(1,2,3-cd)pyrene	0.0171

To calculate the slope factor for a given carcinogenic PNA, the slope factor for BaP for the pertinent route of exposure (oral or inhalation) is multiplied by the TEF for that cPNA.

This relative potency approach contains uncertainties; however, it provides an appropriate method to evaluate the potential adverse health effects of carcinogenic PNAs at this site and is similar to the approach developed for evaluating such compounds as dioxins and furans.

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TOXICITY PROFILE

CHROMIUM VI

Introduction

Chromium is a naturally occurring element in soil, volcanic dust, and gases. Chromium VI, also known as chromate, is one valence state in which chromium is found (others are Chromium 0 and Chromium III). Chromium VI is generally produced by industrial processes including the manufacture of steel and other metal alloys, chrome plating, pigment manufacture, leather tanning, and wood and water treatment. Chromium VI is generally the most toxic of the various chromium valence states. Soluble chromium VI compounds are irritating and corrosive, and their toxic effects are the result of these properties (*ATSDR, 1989*). There is some evidence that chromium VI is reduced in the body in part to chromium III, an essential nutrient (*EPA, 1992a*). The target sites for chromium VI toxicity include the respiratory tract, kidneys, liver, and immune system. Further details of chromium VI toxicity are presented below.

Developmental/Reproductive Toxicity

Data on the developmental or reproductive toxicity of chromium VI following inhalation or oral exposure in humans could not be found in the available literature. Animal studies involving oral exposure found no increase in adverse reproductive effects. Animal studies involving parenteral administration (injection) found increased rates of fetal death, external abnormalities, and weight depression (*ATSDR, 1989*). The relevance of these findings to humans is not known.

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Noncarcinogenic Effects

Acute Toxicity

The acute effects of exposure to chromium in humans generally result from occupational inhalation of dusts or mists. Inhalation exposure to chromium causes dyspnea, cough, pharyngitis, and nasal and generalized respiratory irritation. Immune system effects in the form of anaphylactoid reactions have also been seen. When ingested, chromium VI can cause renal tubular necrosis, gastrointestinal tract irritation and bleeding, nausea, vomiting, hepatic necrosis, and hypersensitivity reactions including dermatitis, urticaria, and angioedema. Dermal exposure can result in dermatitis and deep ulcers (*ATSDR, 1989*).

Chronic Toxicity

Adverse respiratory effects in humans that result from chronic inhalation of various chromium VI compounds include ulceration and perforation of the nasal septum, necrosis and atrophy of the bronchial epithelium, polyps and papillomas of the upper respiratory tract, emphysema, chronic bronchitis, chronic pharyngitis, tracheitis, and pneumonia. Workers chronically exposed via inhalation have developed nephrotoxicity in the form of renal tubular necrosis and hepatic injury with jaundice. Skin lesions including allergic dermatitis and eczema have also resulted from chronic inhalation exposure (*ATSDR, 1989*).

Laboratory animals subchronically exposed orally and intraperitoneally have shown signs of neurotoxicity including decreased motor activity and neuronal degeneration in the cerebral cortex, suggesting that the brain may also be a target for chronic chromium VI toxicity in humans (*ATSDR, 1989*). Few data on chronic oral toxicity of chromium VI in humans were located in the available scientific literature.

The EPA-reported chronic oral reference dose (cRfD) for chromium VI is 5 x 10^{-3} mg/kg/day. This value was based on the work of MacKenzie et al. (*1958*), who observed no adverse effects on appearance, weight gain, food consumption, blood, or other tissues in male and female Sprague Dawley rats when administered chromium VI as potassium dichromate in drinking water for one year. The no-observed adverse effect level (NOAEL) calculated for this study was 2.4 mg/kg/day. An uncertainty factor (UF) of 500 was applied to the NOAEL to calculate the cRfD on the basis of expected interhuman and interspecies variability in toxicity in the absence of specific data and to compensate for reliance on a study with an exposure duration of less than lifetime. The EPA-reported subchronic oral reference dose (sRfD) is 2 x 10^{-2} mg/kg/day and was based on the same study as the cRfD. The UF for the sRfD is 100; no other details were reported. The study on which the RfDs were based is rated low in confidence by the EPA because of the small number of animals tested, the small number of parameters measured, and the lack of a toxic effect at the highest dose tested; confidence in the database is also low because the supporting studies were of low quality and because the teratogenic and reproductive endpoints have not been well studied (*EPA, 1992b*). The EPA therefore assigned a low confidence rating to the oral RfD. The EPA has withdrawn the inhalation reference concentration (RfC) for chromium VI, pending review of the data (*EPA, 1992a,b*).

Carcinogenic Effects

Epidemiological studies indicate an increased respiratory cancer risk from occupational exposure to chromium via inhalation. Animal studies support this correlation between chromium exposure and lung tumors. The EPA has calculated an inhalation unit risk value of 1.2 x 10^{-2} micrograms per cubic meter ($\mu g/m^3$), which is

equivalent to an inhalation slope factor of 42 $(mg/kg/day)^{-1}$ (*EPA, 1992a*). This value was based on the work of Mancuso (*1975*), who observed an increased incidence of lung cancer in workers chronically exposed to chromium VI via inhalation. The EPA has not yet reported an oral slope factor; a value is currently under review. The EPA has classified chromium VI as a Group A carcinogen (known human carcinogen) on the basis of sufficient human and animal data (*EPA, 1992b*). In general, the positive genotoxicity results for chromium VI support the carcinogenicity data in human and animal studies.

In the absence of an EPA-determined oral SF, the PHEE used 4.2×10^{-1} $(mg/kg/day)^{-1}$ as the oral SF. This value was established by the Office of Environmental Health Hazard Assessment of the California Environmental Protection Agency (*Cal-EPA, 1992*).

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TOXICITY PROFILE

LEAD

Introduction

Lead is a bluish white or silvery gray metal whose prevalence in the environment results from both naturally occurring and anthropogenic sources. Lead occurs naturally in the earth's crust and soil, rarely in the elemental state, and usually in a number of ores. Natural sources that contribute to airborne lead include silicate dusts, volcanic halogen aerosols, forest fires, sea salt, meteoric matter, and radon decay. Anthropogenic sources are the mining, smelting, manufacture, and refinement of lead, coal-fired power plants, batteries, vehicle exhaust, waste oil, and iron and steel manufacture (*HSDB, 1991*). This profile assesses the toxicity of metallic lead and inorganic compounds, not organic lead compounds. Lead primarily affects the blood, cardiovascular system, central and peripheral nervous system, and kidneys. Further details of lead toxicity are presented below.

Developmental/Reproductive Toxicity

Evidence suggests an association between human fetal lead exposure and the subsequent retardation of mental development at blood-lead levels as low as 10 micrograms per deciliter (µg/dl) (*Bellinger et al., 1987*). Postnatal effects in the offspring of orally exposed animals include delayed air righting reflex and time to eye opening, decreased visual acuity and evoked responses, and learning and behavioral disabilities (*Kishi et al., 1983; Winneke et al., 1977; Winneke, 1980; Bushnell and Bowman, 1979; Laughlin et al., 1983; Cooper et al., 1980; Fox and Wright, 1982; Fox et al., 1977; Impelman et al., 1982*).

Toxicity tests with laboratory animals have shown that adverse effects on reproductive function in both males and females can occur from oral exposure to inorganic lead (*Rom, 1980*). Effects include ovarian changes in rhesus monkeys, delay of sexual maturity and decrease in clutch size in hens, and prostate hyperplasia and reduction of testicular weight in male rats. The oral administration of inorganic lead to adult rats of both sexes appears to produce synergistic effects, i.e., combined toxic effects on reproduction and on the offspring that are greater than those produced by treatment of either sex alone (*Stowe and Goyer, 1971*). In humans, decreased fertility and abnormal sperm were found in inhalation-exposed male workers with blood-lead levels as low as 53 µg/dl (*Lancranjan et al., 1975*).

Noncarcinogenic Effects

Acute Toxicity

Acute oral or inhalation exposure to inorganic lead can result in encephalopathy (degenerative brain disease) characterized by headache and drowsiness and at higher doses by coma, convulsions, and possibly death. Acute lead encephalopathy often results in permanent, residual, neuropsychologic impairment characterized by reduced intelligence and behavior changes. Acute lead exposure via ingestion can also result in the Fanconi Syndrome characterized by injury to the renal tubules of the kidneys and leakage of glucose, amino acids, and phosphates into the urine (*ATSDR, 1990*).

Chronic/Subchronic Toxicity

The main targets of chronic oral or inhalation exposure to inorganic lead in humans are the red blood cells and their precursors, the cardiovascular system, the central and peripheral nervous system, and the kidneys. Anemia can be among the most sensitive manifestations of the hematologic toxicity of lead, resulting primarily from the

lead-induced inhibition of enzymes involved in heme biosynthesis (*Hernberg and Nykänen, 1970*). Increased blood pressure has been associated with blood-lead concentrations ranging from 30 to 40 µg/dl to as low as 7 µg/dl (*EPA, 1986*). This relationship appears most strongly in middle-aged white males (aged 40 to 59) although a considerable degree of uncertainty surrounds the statistical analyses of the studies giving rise to this conclusion. Chronic oral or inhalation exposure to inorganic lead can affect the peripheral nervous system, causing segmental demyelination at high doses, and a slowing of motor nerve conduction velocity at lower doses (*Landrigan et al., 1976*). In the central nervous system, chronic oral or inhalation exposure to inorganic lead has been shown to cause subtle but apparently irreversible deficits in intelligence and behavior (*Needleman et al., 1979; Winneke et al., 1981; Yule et al., 1981*). On the basis of these and other data, the EPA (*1986*) concluded that lead causes subtle but irreversible damage to the central nervous system in children at blood levels below 50 µg/dl, possibly as low as 10 µg/dl.

To date, the EPA has not developed oral or inhalation reference doses (RfDs) for inorganic lead (*EPA, 1992*). Inorganic lead is evaluated differently than other compounds based on its complex pharmacokinetics in the body (*EPA, 1992*).

Carcinogenic Effects

The International Agency for Research on Cancer (IARC) performed an assessment of the carcinogenicity of inorganic lead compounds in humans and found that there is inadequate evidence that lead is a human carcinogen although it has been shown to be a potent carcinogen in several animal species (*Kanzantsis, 1981*). Renal tumors have been found in rats, mice, and hamsters orally exposed to lead (*Boyland et al., 1962; Zollinger, 1953; Van Esch and Kroes, 1969*). Tumors have also been found to develop in

the brain, spinal column, adrenals, prostate, and mammary glands as a result of oral exposure (*Baldwin et al., 1964; Zawirska and Medras, 1972*). Studies of rats exposed to 500 to 2,000 mg/kg of lead in the diet found tumors; however, it should be noted that these high doses can also cause noncarcinogenic effects in experimental animals. The EPA has not established oral or inhalation slope factors for lead, which it classifies as a Group B2 chemical, a probable human carcinogen (*EPA, 1992*). Instead, two models developed by EPA and Cal-EPA were used to predict blood-lead levels resulting from potential exposures to lead at Sites IR-9, IR-6, and IR-10 (*EPA, 1990; Cal-EPA, 1992*).

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TOXICITY PROFILE

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THALLIUM

Introduction

Thallium is a soft, odorless, bluish-white metal that naturally occurs in trace amounts in the earth's crust. It is found as a pure element, in alloys with other metals, or as inorganic salts. The thallous state (I) is more commonly found in the environment than the thallic state (III). Industrial uses of thallium, mostly in the electronic industry, increase the potential for human exposure to thallium in the environment (*ATSDR, 1990*). Target sites of thallium toxicity include the respiratory, cardiovascular, gastrointestinal and nervous systems, and the liver and kidneys. Further details of thallium toxicity are discussed below.

Developmental/Reproductive Effects

Children exposed to thallium *in utero* were found to have no increase in the incidence of birth defects (*Dolgner et al., 1983*). Other data regarding potential developmental effects of thallium on humans were not located in the available scientific literature. Laboratory animals exposed to thallium *in utero* have exhibited fetal growth retardation (chickens and rats), teratogenicity in the form of achondroplasia (dwarfism, in chickens), hydronephrosis and vertebral abnormalities (rats), and functional neurological effects in the form of learning impairment (rats; *ATSDR, 1990; EPA, 1980*).

No data regarding the reproductive effects of thallium on humans were located in the scientific literature; however, animal data suggest that there may be reproductive effects in males. Male rats orally exposed to thallium exhibited decreased sperm motility and histological changes in the testes (*Formigli et al., 1986*).

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Noncarcinogenic Effects

Acute Toxicity

Acute ingestion of large doses (up to 1 gram) of thallium sulfate has caused death due to cardiac or respiratory failure in humans. Acute oral exposure in humans has resulted in lung damage (alveolar damage, pulmonary edema, and bronchopneumonia), cardiac damage (myocardial damage and abnormalities in cardiac rate and rhythm), renal damage (tubular necrosis), liver damage, and gastrointestinal effects including abdominal pain, vomiting, diarrhea, and constipation (*ATSDR, 1990*). Dermal effects (hair loss) and neurological effects (axonal degeneration of cranial and peripheral nerves) have also been reported following acute oral exposure (*ATSDR, 1990*).

Chronic Toxicity

Data regarding chronic exposure to thallium in humans are limited. Workers who were chronically exposed via inhalation developed peripheral nervous system effects including paresthesias, numbness of fingers and toes, muscle cramps, and impaired peripheral nerve conduction (*Ludolph et al., 1986*). However, inadequacies in study design render these results suggestive rather than conclusive. Animal studies of chronic oral exposure to thallium demonstrated degeneration of nerve fibers and myelin sheaths (*Manzo et al., 1983*). These animal studies support the findings of neurological damage in chronically exposed humans.

The EPA-reported chronic oral reference dose (cRfD) for thallic oxide (the most toxic of the thallium compounds listed on EPA's *Integrated Risk Information System*, or IRIS) is 7×10^{-5} mg/kg/day. This value is based on a 90-day subchronic study in which rats were administered thallic sulfate via gavage. Adverse effects to the liver or the blood were not observed at a reported no-observed adverse effect level (NOAEL) of 0.22 mg/kg/day. The NOAEL was divided by an uncertainty factor of 3000, which

consisted of 10 for interspecies conversion, 10 for extrapolation from a subchronic to a chronic exposure duration, and 10 to protect sensitive human subpopulations. A modifying factor of 3 was also used (details not reported). The subchronic reference dose (sRfD), based on the same study, is 7×10^{-4} mg/kg/day. The EPA has not established an inhalation RfD for thallium or its salts (*EPA, 1992a*).

Carcinogenic Effects

No studies were located in the available scientific literature regarding potential carcinogenic effects of thallium on humans or animals. Studies of genotoxicity in humans were not located; however, animal studies indicate that thallium may be genotoxic (*ATSDR, 1990*). The EPA has not determined a weight-of-evidence classification, or oral or inhalation slope factors (SFs) for thallium. Thallic oxide is classified as a Group D chemical, that is, not classifiable as to human carcinogenicity, and no SFs are available (*EPA, 1992a,b*).

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EXHIBIT D

241-6th Street Site
San Francisco, California

The Cap has been constructed as outlined in this exhibit

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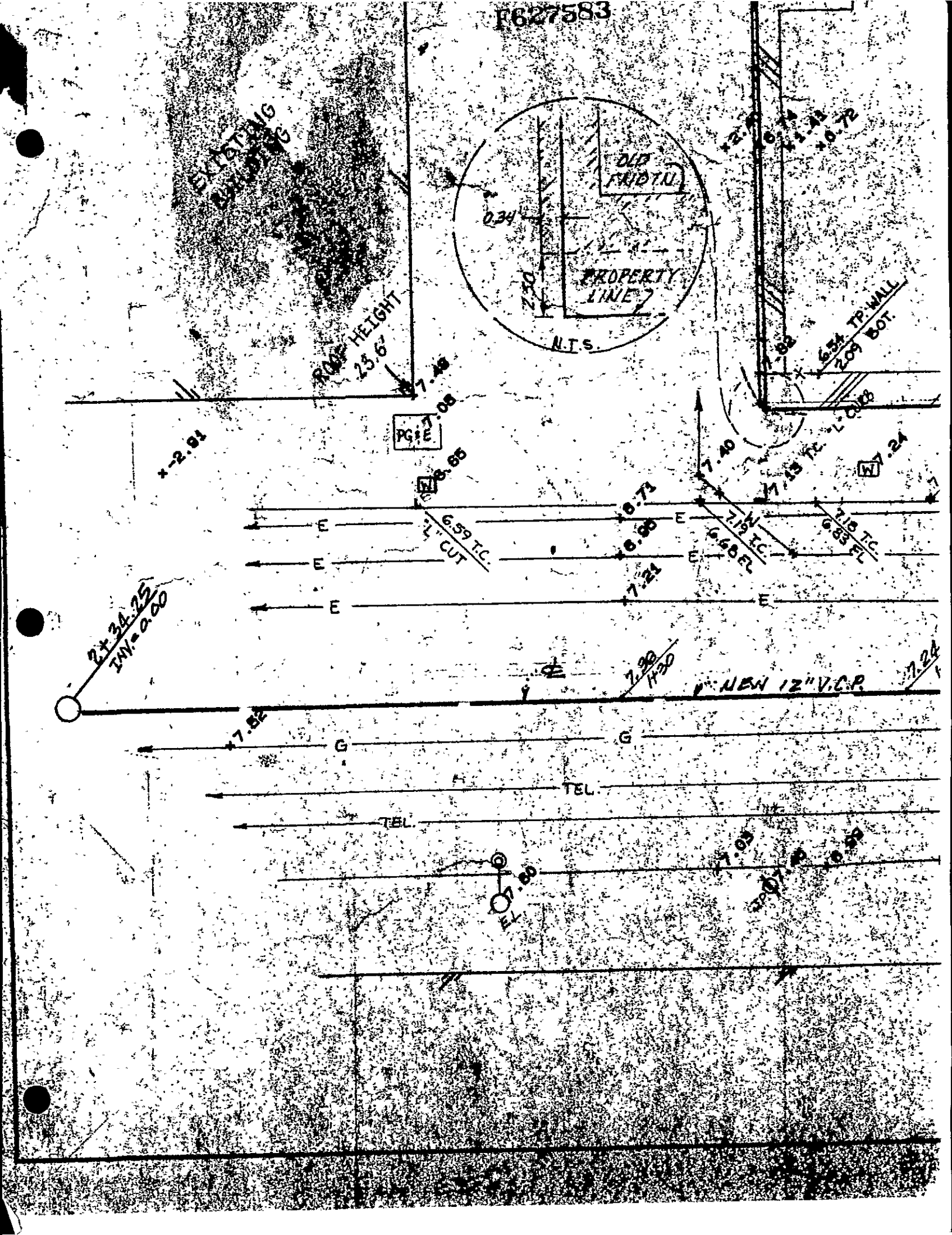
EXISTING BUILDING
ROOF HEIGHT 23.6'
OLD FMID T.U.
0.34
7.30
PROPERTY LINE
N.T.S.
+2.7
6.1
+1.12
+0.72
6.54 TP. WALL
2.09 BOT.
7.48
7.08
PG&E
x-2.81
8.66
W
6.59 T.C.
4" CUT
8.71
8.66
7.40
7.13 T.C.
7.24
W
7.12 T.C.
6.68 EL
7.18 T.C.
6.83 EL
E
7.21
2+34.25
INV.=0.60
7.30
H30
8" NEW 12" V.C.P.
7.24
7.52
G
TEL.
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7.80
EL
7.09
6.98
TP 7.49

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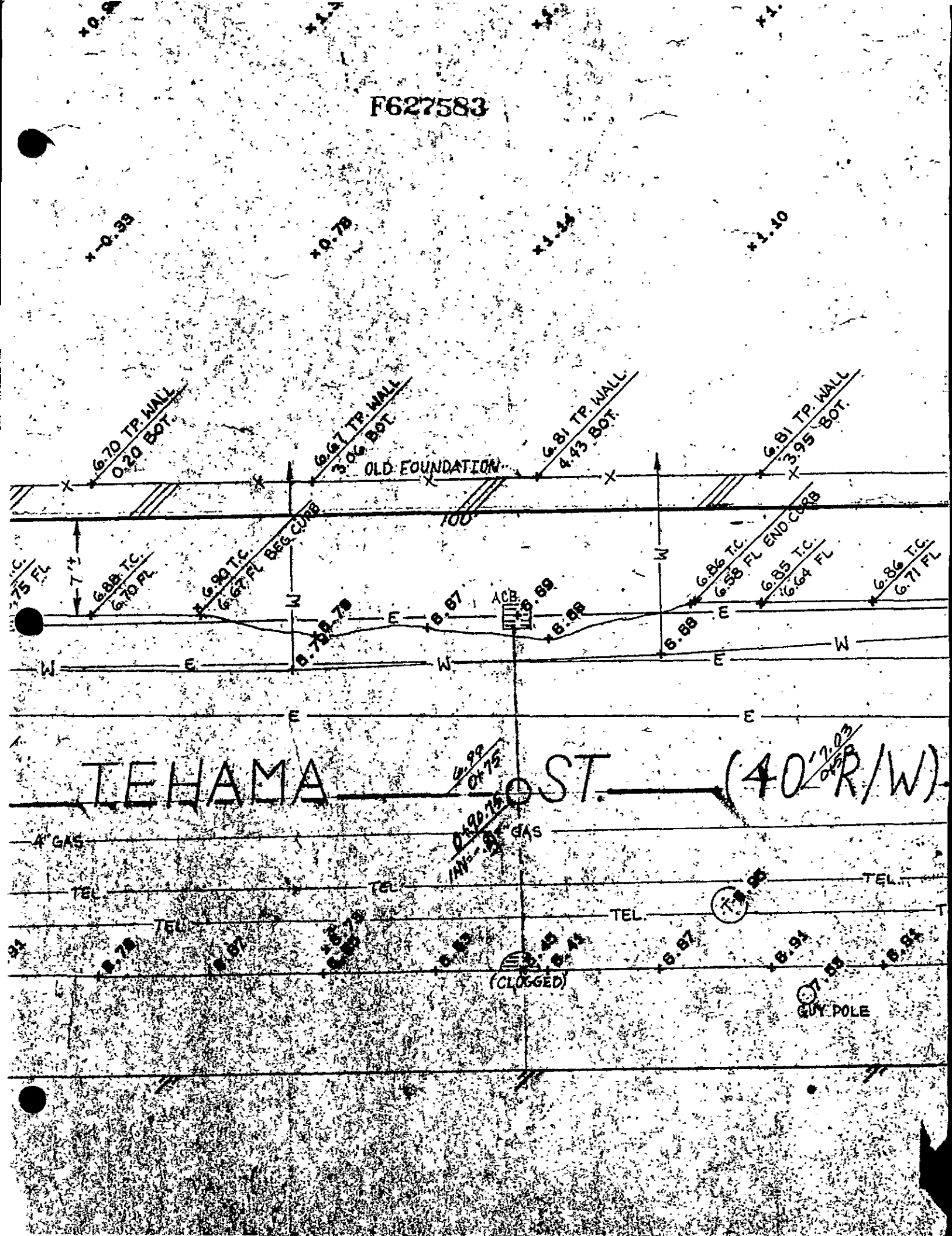
6.70 TP. WALL
0.20 BOT.
6.67 TP. WALL
3.06 BOT.
OLD FOUNDATION
6.81 TP. WALL
4.43 BOT.
6.81 TP. WALL
3.95 BOT.
100
6.88 T.C.
6.70 FL
6.90 T.C.
6.67 FL BEG. CURB
6.86 T.C.
6.58 FL END CURB
6.85 T.C.
6.64 FL
6.86 T.C.
6.71 FL
ACB
6.89
6.88
6.87
6.88
W
E
TEHAMA ST (40' R/W)
4" GAS
TEL
(CLOGGED)
GUY POLE